

# Northumbria Research Link

Citation: Ansdell, Paul and Dekerle, Jeanne (2017) Sodium bicarbonate supplementation delays neuromuscular fatigue without changes in performance outcomes during a basketball match simulation protocol. Journal of Strength and Conditioning Research. ISSN 1064-8011 (In Press)

Published by: Lippincott Williams & Wilkins

URL: <https://doi.org/10.1519/JSC.0000000000002233>  
<<https://doi.org/10.1519/JSC.0000000000002233>>

This version was downloaded from Northumbria Research Link:  
<http://nrl.northumbria.ac.uk/39761/>

Northumbria University has developed Northumbria Research Link (NRL) to enable users to access the University's research output. Copyright © and moral rights for items on NRL are retained by the individual author(s) and/or other copyright owners. Single copies of full items can be reproduced, displayed or performed, and given to third parties in any format or medium for personal research or study, educational, or not-for-profit purposes without prior permission or charge, provided the authors, title and full bibliographic details are given, as well as a hyperlink and/or URL to the original metadata page. The content must not be changed in any way. Full items must not be sold commercially in any format or medium without formal permission of the copyright holder. The full policy is available online: <http://nrl.northumbria.ac.uk/policies.html>

This document may differ from the final, published version of the research and has been made available online in accordance with publisher policies. To read and/or cite from the published version of the research, please visit the publisher's website (a subscription may be required.)

[www.northumbria.ac.uk/nrl](http://www.northumbria.ac.uk/nrl)



## TITLE PAGE

Sodium bicarbonate supplementation delays neuromuscular fatigue without changes in

### Authors

Paul Ansdell<sup>1,2</sup>, Jeanne Dekerle<sup>1</sup>

Sport, Exercise Science And Medicine (SESAME), University of Brighton, Eastbourne, United Kingdom.

Faculty of Health and Life Sciences, Department of Sport, Exercise & Rehabilitation, Northumbria University, Newcastle, United Kingdom

### Address for correspondence:

Paul Ansdell

Faculty of Health and Life Sciences

Department of Sport, Exercise & Rehabilitation

Northumbria University

Newcastle upon Tyne

NE1 8ST

performance outcomes during a basketball match simulation protocol

UK

**Email :** paul.ansdell@northumbria.ac.uk

**Phone:** +44 (0)191 243 7018

**Fax:** +44 (0)191 227 4713

**Disclosure statement of funding received for this work:** none

**Conflict of interest:** none

## ABSTRACT

**Purpose:** To investigate the development of neuromuscular fatigue during a basketball game simulation and ascertain whether sodium bicarbonate ( $\text{NaHCO}_3$ ) supplementation attenuates any neuromuscular fatigue that persists. **Methods:** Ten participants ingested  $0.2 \text{ g.kg}^{-1}$  of  $\text{NaHCO}_3$  (or an equimolar placebo dosage of sodium chloride [ $\text{NaCl}$ ]) 90 and 60 minutes prior to commencing a basketball game simulation (ALK-T vs PLA-T). Isometric maximal voluntary contractions of the knee extensors (MVIC) and potentiated high (100 Hz) and low (10 Hz) frequency doublet twitches were recorded before and after each match quarter for both trials. In addition, 15 m sprint times and layup completion (%) were recorded during each quarter. **Results:** MVIC, 100 and 10 Hz twitch forces declined progressively in both trials ( $P < 0.05$ ) with a less pronounced decrease in MVIC during ALK-T ( $P < 0.01$ ). Both 100 and 10 Hz twitch forces were also significantly greater in ALK-T ( $P < 0.05$ ). 15 m sprint time increased over the course of both trials ( $\sim 2\%$ ,  $P < 0.01$ ); however, no significant condition or time effect was found for layup completion ( $P > 0.05$ ). **Conclusion:** A basketball simulation protocol induces a substantial amount of neuromuscular (reduction in knee extensor MVICs) and peripheral fatigue with a concomitant increase in 15 m sprint time over the protocol.  $\text{NaHCO}_3$  supplementation attenuated the rate of fatigue development by protecting contractile elements of the muscle fibres. **Practical Applications:** This study provides coaches with information about the magnitude of fatigue induced by a simulated basketball game, and provides evidence of the efficacy of  $\text{NaHCO}_3$  in attenuating fatigue.

## KEY WORDS

Alkalosis; muscular fatigue; peripheral fatigue; team sports

## INTRODUCTION

Basketball matches are characterised by a large volume of short duration, high intensity movements as shown via time-motion analysis (25). Simulated games can also raise mean oxygen uptake ( $\dot{V}O_2$ ) and heart rate (HR) values to approximately 65% and 85% of their maximum, respectively (25, 28). Due to the elevated metabolic demand of a basketball game, a build-up of deleterious metabolites (i.e.  $H^+$ , Pi) may reduce force producing capacity of the working muscles (2). This is deemed neuromuscular fatigue, and is defined in the present work as any transient, exercise-induced reduction in muscular force generating capacity (42), with underpinning mechanisms of peripheral or central origins. Previous research has shown that explosive power and sprint ability is reduced following basketball-related activity (9). However, to our knowledge, no study has yet investigated the time course of various sites of neuromuscular fatigue during a simulated basketball match, and the efficacy of a potentially ergogenic supplement in ameliorating the aforementioned fatigue by reducing metabolite accumulation.

As a result of high intensity exercise (such as basketball), extra and intra-cellular ionic concentrations are altered within the muscles, causing reduced contractile performance. For a complete review of the processes contributing to peripheral fatigue see Allen et al (2). Examples of factors involved in impairment within the muscular contractile apparatus are reduced intracellular potassium ion ( $K^+$ ) concentrations caused by efflux into the interstitial spaces, resulting in extracellular accumulation (19). This negatively affects the capacity of the sarcolemma to propagate action potentials (27). This potentially occurs as  $Na^+$ ,  $K^+$  ATPase activity is inhibited by the increased presence of hydrogen ions ( $H^+$ ) (acidosis).

Similarly, acidosis inhibits myofibril ATPase activity, leading to reduced calcium ion ( $\text{Ca}^{2+}$ ) reuptake to the sarcoplasmic reticulum (SR) and consequently less  $\text{Ca}^{2+}$  released from the SR when prompted by an action potential (7, 22).

In this study, contractile function was measured using potentiated, electrically-evoked paired twitches at two different frequencies (10 and 100 Hz) in an attempt to refine the sites of peripheral fatigue development. The mechanical response (twitch) to low frequency doublets (10 Hz) has been shown to be modulated by the extent of  $\text{Ca}^{2+}$  release from the SR (19, 21). High frequency doublets (100 Hz), and their respective twitch amplitude, have been shown not to be affected by moderate decreases in  $\text{Ca}^{2+}$ . Therefore decreases in high frequency twitch amplitude reflect attenuated action potential propagation caused by extracellular  $\text{K}^+$  accumulation (21). The ratio of low:high frequency twitch forces gives detail about the aetiology of contractile decline during a fatiguing task (27).

The role acidosis plays in the development of neuromuscular fatigue during high intensity exercise (such as basketball) remains under debate (10, 44). However, it is generally agreed that athletes who perform high intensity exercise (such as basketball players) would likely benefit from  $\text{NaHCO}_3$  supplementation (8) as it attenuates the aforementioned negative effects of acidosis. For instance, a reduction in extracellular accumulation of  $\text{K}^+$  during exhaustive exercise has been evidenced following  $\text{NaHCO}_3$  supplementation (40).  $\text{NaHCO}_3$  supplementation also attenuates the inhibiting effects of  $\text{H}^+$  by increasing the intra – extracellular pH gradient. This allows for greater efflux of deleterious metabolites outside the muscle cells and attenuates their harmful effects on the contractile function (26).

NaHCO<sub>3</sub> has been shown to enhance high-intensity performance (5, 6) and delay the development of neuromuscular fatigue during a fatiguing task (38). The ergogenic effects found in the aforementioned laboratory-based studies give rationale for the investigation of NaHCO<sub>3</sub> as an ergogenic aid during high-intensity team sport activity such as basketball. Interestingly, only a limited amount of studies have investigated the effect of NaHCO<sub>3</sub> supplementation on performance outcomes during simulated game based protocols (1, 23, 31, 34), and neuromuscular function has never been assessed throughout a simulated basketball match. Afman et al (1) recently found a beneficial effect of NaHCO<sub>3</sub> supplementation on 15m sprint times, but not layup completions, during a modified Loughborough Intermittent Sprint Test (LIST), which was validated to replicate the demands of a 40-min basketball game. Therefore, the present study aims to investigate the development of neuromuscular fatigue and more specifically, the peripheral mechanisms during a basketball game simulation. The study also aims to ascertain whether NaHCO<sub>3</sub> supplementation attenuates this development. It was hypothesised that there would be a significant decline in both voluntary force generating capacity of the knee extensors and the amplitude of evoked paired-twitches. It was hypothesised that this decrease in contractile function would lead to faster 15-m sprint times throughout the protocol and with smaller declines in the supplement (ALK-T) compared to the placebo (PLA-T) trial.

## **METHODS**

### ***Experimental Approach to the Problem***

Participants visited the laboratories on three separate occasions. Three and seven days separated familiarisation and 1<sup>st</sup> fatiguing trial, and 1<sup>st</sup> and 2<sup>nd</sup> fatiguing trials respectively, to ensure full washout of the supplement/placebo (5). Familiarisation involved a neuromuscular function

assessment performed on an isokinetic dynamometer, followed by one block of the modified LIST protocol. For the two experimental trials, participants performed four blocks of the modified LIST with neuromuscular assessment prior to, and following each block of the LIST (1). Participants were asked to avoid consuming any stimulants or alcohol, and to replicate food intake during a 24-hour period before testing. The study was a double-blind crossover design with exposure to supplements randomized and counterbalanced. Each participant received extensive information, and signed an informed consent form and medical questionnaire after they had the opportunity to ask any questions to researchers. The protocol was approved by the University Ethics committee and adhered to the Declaration of Helsinki.

### ***Subjects***

Ten healthy and active male basketball players volunteered to take part in the study (age  $21 \pm 1$  years; height:  $182 \pm 5$  cm; weight:  $81.5 \pm 8$  kg). All participants had over 4 years of competitive basketball experience.

### ***Procedures***

#### ***Neuromuscular Function Assessment***

For the neuromuscular assessment of the right knee extensors, participants sat on the Con-Trex Multi-Joint system (Con-Trex, Dubendorf, Switzerland) as per the published reliability study (30) (~85° hip angle; distal dynamometer's shin pad attached 2–3 cm proximal to the lateral malleolus with a strap around the shank; straps were fastened and locked across chest and pelvis; movement resisting pad over the mid-thigh of the contracting leg). Knee angle was kept at 90° for all maximal voluntary isometric contractions (MVICs) and twitches.



Torque measurement was corrected to take gravity effect into account. Participants were instructed to cross their arms across their chest and were provided with visual feedback of force during the protocol.

A 48 mm<sup>2</sup> self-adhesive cathode electrode (CF3200, Nidd Valley Medical Ltd, Harrogate, UK) was placed directly over the femoral nerve in the femoral triangle with the anode placed directly onto the greater trochanter of the femur (Prottens, Bio Protech Inc, Korea).

Percutaneous electrical stimulation was delivered by a constant-current stimulator (DS7A, Digitimer, Letchworth Garden City, Great Britain). Stimulations were triggered manually using a PowerLab 15T (Model ML818, AdInstruments Pty Ltd, Dunedin, New Zealand) and force production was recorded using LabChart 7 software (AdInstruments Pty Ltd, Dunedin, New Zealand). Sprint times (15-m) were recorded using wireless electronic timing gates (TC Timing System, Brower, Utah, USA). Participants began each sprint from a standing start 10cm behind the timing gates (see figure 1).

#### *Familiarisation Session*

Single electrical 200µs impulses were delivered to the right femoral nerve via the surface electrode. Percutaneous single stimuli were delivered at 10 mA increasing by 10mA until a plateau in twitch force amplitude was reached. This intensity was increased to 130%, to ensure supramaximal stimulations were delivered (mean intensity: 170 ± 35 mA). This process was repeated before each experimental visit. The MVIC familiarisation protocol then consisted of 2 and then 3 × 5-s voluntary contractions performed at 50% and 75% of maximal subjective effort, respectively. The participants then performed 3 × 5-s MVICs. Each maximal contraction was followed by two doublet stimulations (100 Hz and 10 Hz) in 1-s intervals.

129

## 130 *Experimental Protocol*

131 Neuromuscular baseline tests were performed followed by a short standardised warm up (a  
132 4length jog of the basketball court). After baseline and warm ups, participants completed four  
133 blocks of 11 repetitions of the modified LIST shown in figure 1 (1), meaning 11 sprints and

layups were performed per quarter. Participants had 5 minutes rest between quarters, in  
which neuromuscular fatigue assessment was performed. Three 5 s MVICs with 60 s rest  
between were performed with two doublet stimulation s (10 Hz and 100 Hz) following the  
contractions in 1 s intervals. Due to the time taken to move from basketball court to the  
dynamometer following each quarter, the timing of the first MVIC was standardised to 75 s.

FIGURE 1 HERE

### *Supplement*

Participants arrived 90 minutes prior to commencement of the protocol in order to consume  
the first half of either the supplement or placebo;  $\text{NaHCO}_3$  was delivered in two separate  
dosages of 0.2 g.kg<sup>-1</sup> with the second dosage consumed 60 minutes prior.  $\text{NaHCO}_3$  was  
dissolved in 500 ml of low-calorie-free cordial each, totalling 0.4 g.kg<sup>-1</sup>. Sodium  
chloride (placebo) was composed of two 0.138 g.kg<sup>-1</sup> dosages dissolved in 500 ml water and  
cordial each (equimolar amount of sodium to account for alterations in  $\text{Na}^+$  handling; for  
134 more details, see (20)). The same amount of supplement/placebo was consumed 60 minutes  
135 prior to exercise. A similar ingestion protocol has been shown to benefit prolonged intermittent  
136 activity with no reported incidences of gastrointestinal disturbances following  $\text{NaHCO}_3$   
137 supplementation, as did the present study (5).

## *Data Analysis*

The maximum 500-ms value was recorded as maximal force for each MVIC plateau, and the peak twitch amplitude was computed for each doublet stimulation. The greatest value over each set of three MVICs and twitches was subsequently recorded for each time point.

Coefficient of variations between the 3 measures were  $2.6 \pm 2.0\%$  for MVIC,  $3.5 \pm 2.7\%$  for 100 Hz twitch, and  $3.0 \pm 2.2\%$  for 10 Hz twitch. Each 15 m sprint time was recorded in seconds (s). Successful completions for the layups were expressed as a percentage of total number of attempts per quarter (out of 11).

## *Statistical Analysis*

Normal distributions were verified (Kolmogorov-Smirnov test) and one-way ( $1 \times 5$ ) repeated measures ANOVAs were run to assess the change in neuromuscular variables (MVC, 100Hz, 10 Hz twitches) and quantify the magnitude of fatigue elicited over the course of the placebo trial (Baseline, Q1, Q2, Q3, Q4). A two-way ( $2 \times 5$ ) repeated measures ANOVAs was performed to test for between condition (ALK-T vs PLA-T) and time differences (Baseline, Q1, Q2, Q3, Q4). If sphericity assumption was violated (Mauchly's test) then Frattios were adjusted according to the Greenhouse-Geisser procedure. Significant effects of ANOVAs were followed up using the Bonferroni-corrected pairwise post hoc test.

Significance was accepted at  $P \leq 0.05$  and all data is presented as mean  $\pm$  standard deviation (SD). All statistical analyses were performed using SPSS (version 20, Chicago, USA).

## RESULTS

TABLE 1 HERE

FIGURE 2 HERE

MVIC force ( $F_{(4,36)} = 42.0$ ,  $P < 0.01$ ), decreased significantly over time but with no significant difference between conditions ( $P > 0.05$ ) (Table 1 and Figure 2). The loss of MVIC was less pronounced during ALK-T as shown by the significant time  $\times$  condition interaction ( $F_{(4,36)} = 6.88$ ,  $P < 0.01$ ). However, post-hoc tests did not reveal a significant difference between trials at any time points ( $P > 0.05$ ). The one way ANOVA showed that during PLA-T, the decrement in MVIC ( $F_{(4,36)} = 36.9$ ,  $P < 0.01$ ) was progressive from baseline to the 3<sup>rd</sup> quarter ( $P < 0.05$ ), with a plateau occurring thereafter ( $P > 0.05$ ).

100 Hz twitch ( $F_{(4,36)} = 20.25$ ,  $P < 0.01$ ) and 10 Hz twitch ( $F_{(4,36)} = 24.3$ ,  $P < 0.01$ ) also decreased significantly over time. No time  $\times$  condition interaction effect was found for either evoked twitches (100 Hz:  $F_{(4,36)} = 0.76$ ,  $P = 0.56$ ; 10 Hz:  $F_{(4,36)} = 1.30$ ,  $P = 0.29$ ). 100 Hz and 10 Hz evoked twitch forces were both greater throughout the protocol in ALK-T (condition effect: 100 Hz:  $F_{(1,9)} = 11.8$ ,  $P < 0.01$ ; 10 Hz:  $F_{(1,9)} = 8.77$ ,  $P < 0.05$ ). The one way ANOVA showed that during PLA-T, 100 and 10 Hz twitches were not different from baseline ( $P > 0.05$ ) until after the second quarter from which time point a reduction was significant ( $P < 0.05$ ). No time or condition effect was observed for 10:100 Hz twitches ratio ( $P > 0.05$ ).

No condition or interaction effect were found for either of the performance variables ( $P > 0.05$ ) but the 15-m sprint times became significantly slower over both trials (time effect:  $F_{(3,27)} = 9.39$ ,  $P < 0.01$ ). The participants' sprints in both trials were systematically slower from one

quarter to the next ( $P < 0.05$ , Table 1). When comparing first vs last quarter sprint times, both ALK-T and PLA-T were significantly longer (ALK-T: -1.7%,  $F_{(3,7)} = 4.3$ ,  $P < 0.01$ ; PLA-T -2.4%,  $F_{(3,7)} = 9.3$ ,  $P < 0.05$ ).

## DISCUSSION

To our knowledge this is the first study reporting development of neuromuscular fatigue during simulation of a basketball match. Maximal force production of the knee extensors (MVIC) during PLA-T reduced throughout the first three quarters of the simulated match (Figure 2; Table 1; ~5% loss per quarter) with no further reduction in the final quarter. Peripheral fatigue was evident from the 2<sup>nd</sup> quarter of the protocol with disturbances of contractile properties. The ~15% reduction in MVIC torque recorded post 3<sup>rd</sup> and 4<sup>th</sup> quarter in this study is similar to the ~15% reduction reported after a 60-min squash match (12), and within the ~11% (11, 13, 29) to ~20% range (16, 17) reported for laboratory based studies investigating repeated sprint activity (4-to 10-s sprints, 8-12 repetitions, 10- to 30-s passive recovery).

To our knowledge, this is also the first study applying femoral nerve stimulations to assess mechanisms of peripheral fatigue during a basketball game simulation. The ~15% reductions in evoked twitch forces from baseline for both doublet stimulations are similar to those previously reported in laboratory-based studies following repeated sprint exercise (9-15%; (29, 32)). In the present study, changes in the several mechanisms of contractile function impairment seem to adopt a similar time course with a decrease after the 2<sup>nd</sup> quarter, and no further decrease thereafter (apart from one occurrence: 10 Hz twitch between quarter 2 and 3,  $P = 0.01$ ). Perrey et al (29) found decreases of 15% in low frequency (20 Hz) twitch forces but

of only 8% in the high frequency (80 Hz) twitch forces following repeated sprints. Their decreased ratio of low:high frequency evoked twitches (−9%) suggested that muscle fibre excitability was the predominant cause for the impairment of the contractile function. In contrast, the present study found decreases of similar extent in low and high frequency evoked twitch forces, suggesting that a basketball simulation protocol affects both excitation and contraction mechanisms to a similar degree.

15-m sprint times increased by ~2% in PLA-T following the basketball simulation protocol, compared to Afman et al (1) who reported a ~5% increase during the placebo trial of the modified LIST. This lies within the 2-10% decrease in sprint times of short distances ( $\leq 20$  m) typically reported for team sport activities (3, 4, 18, 24). These reductions in running performance are greater than losses in ‘pure’ strength measurements such as MVICs mentioned earlier (~10-20%). This could be explained by a possible change in sprint mechanics in a fatigued state, affecting speed production as a consequence (33). For instance, in the present study, participants were tightly secured on the dynamometer to avoid any extra bodily movements other than the knee extensors, so that MVIC forces could not be affected by a change in technique. Interestingly however, both evoked twitches were significantly greater in ALK-T, demonstrating the protective effect increased extracellular buffering agents have on both potassium and calcium ion-related contractile properties of the muscle fibres of the knee extensors. This protection of the muscle force-generating capacity is further illustrated in the present study by the attenuation in the continuous development of neuromuscular fatigue (MVIC torque) during the protocol under the  $\text{NaHCO}_3$  supplementation.

Several studies have to date reported the effect of  $\text{NaHCO}_3$  on neuromuscular fatigue. This was following submaximal isometric calf muscles contractions (36), a 2-min voluntary knee extension (35), tetanic stimulation (39), and high-intensity repeated sprint cycling (38). In agreement with our findings, all found no condition effect on MVIC forces from pre- to postexercise. In contrast with the present results however, the force decline was similar in both alkalosis and placebo trials (35). The differences in the fatiguing protocols and measurement methods might explain these discrepancies. Our basketball simulation protocol engaged a greater muscle mass and was of longer duration so that a time  $\times$  condition interaction effect was more likely to occur. Stimulations were applied to the posterior tibial nerve to evoke force in the calf muscles in Siegler et al (36), and as suggested by the authors themselves, the relatively low task demand coupled with the small muscle group might have contributed to the lack of pH effect.

The 15-m sprint times were on average 0.2% faster, and MVIC torque 3.3% greater in ALKT (5 measures,  $n = 10$ ). Whilst 7 out of 10 participants recorded lesser decreases in 100 and 10 Hz twitch amplitudes in ALK-T compared to PLA-T, the two-way ANOVA did not depict any interaction effect. A meta-analysis reported for an ergogenic effect of only 1.7% on some performance indicators such as mean power during repeated sprint exercise (8). Several studies also reported a lack of condition effect on 15-m sprint times following ingestion of a buffering agent compared to a placebo (1, 34). Whilst the present study focussed on mechanisms affecting the contractile apparatus, it should be noted that alterations within the CNS may also be responsible for declines in voluntary force. Team sport activity has been shown to induce substantial decreases in voluntary activation of quadriceps muscles (14, 43).  $\text{NaHCO}_3$  may also attenuate afferent feedback associated with metabolite accumulation (37).

Therefore, the ergogenic effects demonstrated in the present study (i.e. attenuated MVIC force reduction) might not be purely due to protection of contractile mechanisms. Furthermore, NaHCO<sub>3</sub> supplementation in the present study was limited by the absence of blood gas measurements; however there is evidence that a similar supplementation protocol to the one used in this study raises [HCO<sub>3</sub><sup>-</sup>] levels by ~5mmol.L<sup>-1</sup> and sustains elevated blood pH and HCO<sub>3</sub><sup>-</sup> during a prolonged intermittent sprint protocol ENREF 17(5). Factors such as time to peak [HCO<sub>3</sub><sup>-</sup>] and [pH] also show high degrees of inter and intra-individual variability (15, 41). Therefore, it is possible that the ergogenic effect seen in the present study may not have been maximal as the ingestion times were standardised to 90 and 60 minutes.

In conclusion, the present study shows a two-phase response in the development of fatigue over time during a simulated basketball match, with an initial early development of neuromuscular and peripheral fatigue after just two quarters of simulated match. Beyond which no further deleterious effect on the neuromuscular function can be seen. This occurred alongside a slowing down of 15-m sprint times while layup scores remained unchanged. The second major finding is that ingestion of sodium bicarbonate 90 and 60 minutes priorexercise attenuates the above-mentioned development of neuromuscular fatigue. Maximal force production can be preserved until the 3<sup>rd</sup> quarter of the match. The supplementation preserved both potassium and calcium ion-related contractile properties of the knee extensors so that a greater muscular force generating capacity was possible in the alkaline condition when twitches were evoked using paired stimulations of the femoral nerve. This could be the reason maximal force production was preserved during the protocol.

The present findings should be interpreted with caution due to a small sample size weakening overall statistical power. For example, there was a condition effect for MVIC torque alongside



a condition  $\times$  time interaction effect, but with no post-hoc difference depicted. This is not uncommon in the literature surrounding  $\text{NaHCO}_3$  supplementation (36, 38). Another limitation in this study refers to the lack of electromyography (EMG) measurements. The intensity for electrical stimulation was therefore based on a plateau of twitch force with increasing current, rather than based on a plateau identified for the compound muscle action potential (M wave). As a result, there is no absolute confidence that all motor units were innervated by the electrical stimulation. However, the mean intensity in the present study (170 mA) is comparable to that of studies using plateaus in twitch and M-wave amplitudes for the determination of stimulation threshold in the knee extensors in similar population (80-170 mA, (12); ~190 mA, (14)).

## **PRACTICAL APPLICATIONS**

A simulated basketball match protocol causes a significant amount of overall and peripheral fatigue from the 1<sup>st</sup> and 2<sup>nd</sup> quarter, respectively, as quantified by neuromuscular assessments. Sprint times are also slower throughout simulated basketball match. The employment of intelligent substitution timings and tactics should be used to negate this effect.

Supplementing two dosages of  $0.3 \text{ g} \cdot \text{kg}^{-1} \text{NaHCO}_3$  90 and 60 minutes prior to a basketball simulated match protocol can significantly delay the rate of development of neuromuscular fatigue by protecting contractile properties of muscle fibres.

## **Acknowledgements**

This research has not received any external financial support. The results of the present study do not constitute endorsement of the product by the authors or the NSCA.

## References

1. Afman G, Garside RM, Dinan N, Gant N, Betts JA, and Williams C. Effect of carbohydrate or sodium bicarbonate ingestion on performance during a validated basketball simulation test. *Int J Sport Nutr Exerc Metab* 24: 632-644, 2014.
2. Allen DG, Lamb GD, and Westerblad H. Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev* 88: 287-332, 2008.
3. Andersson H, Raastad T, Nilsson J, Paulsen G, Garthe I, and Kadi F. Neuromuscular fatigue and recovery in elite female soccer: effects of active recovery. *Med Sci Sports Exerc* 40: 372-380, 2008.
4. Ascensao A, Rebelo A, Oliveira E, Marques F, Pereira L, and Magalhaes J. Biochemical impact of a soccer match - analysis of oxidative stress and muscle damage markers throughout recovery. *Clinical biochemistry* 41: 841-851, 2008.
5. Bishop D and Claudius B. Effects of Induced Metabolic Alkalosis on Prolonged Intermittent-Sprint Performance. *Medicine & Science in Sports & Exercise* 37: 759-767, 2005.
6. Bishop D, Edge J, Davis C, and Goodman C. Induced metabolic alkalosis affects muscle metabolism and repeated-sprint ability. *Med Sci Sports Exerc* 36, 2004.
7. Bruton JD, LÄNnergren J, and Westerblad H. Mechanisms underlying the slow recovery of force after fatigue: importance of intracellular calcium. *Acta Physiologica Scandinavica* 162: 285-293, 1998.
8. Carr AJ, Hopkins WG, and Gore CJ. Effects of Acute Alkalosis and Acidosis on Performance. *Sports Medicine* 41: 801-814, 2011.
9. Castagna C, Impellizzeri FM, Rampinini E, D'Ottavio S, and Manzi V. The Yo-Yo intermittent recovery test in basketball players. *Journal of Science and Medicine in Sport* 11: 202-208, 2008.
10. Fitts RH. The Role of Acidosis in Fatigue: Pro Perspective. *Med Sci Sports Exerc* 48: 2335-2338, 2016.
11. Girard O, Bishop DJ, and Racinais S. Hot conditions improve power output during repeated cycling sprints without modifying neuromuscular fatigue characteristics. *Eur J Appl Physiol* 113: 359-369, 2013.
12. Girard O, Micallef J-P, Noual J, and Millet GP. Alteration of neuromuscular function in squash. *Journal of Science and Medicine in Sport* 13: 172-177, 2010.
13. Goodall S, Charlton K, Howatson G, and Thomas K. Neuromuscular fatigability during repeated-sprint exercise in male athletes. *Med Sci Sports Exerc* 47: 528-536, 2015.
14. Goodall S, Thomas K, Harper LD, Hunter R, Parker P, Stevenson E, West D, Russell M, and Howatson G. The assessment of neuromuscular fatigue during 120 min of simulated soccer exercise. *Eur J Appl Physiol* 117: 687-697, 2017.

- 332 15. Gough LA, Deb SK, Sparks AS, and McNaughton LR. The Reproducibility of Blood  
333 Acid Base Responses in Male Collegiate Athletes Following Individualised Doses of  
334 Sodium Bicarbonate: A Randomised Controlled Crossover Study. *Sports Medicine*:  
335 111, 2017.
- 336 16. Hureau TJ, Ducrocq GP, and Blain GM. Peripheral and Central Fatigue Development  
337 during All-Out Repeated Cycling Sprints. *Med Sci Sports Exerc* 48: 391-401, 2016.
- 338 17. Hureau TJ, Olivier N, Millet GY, Meste O, and Blain GM. Exercise performance is  
339 regulated during repeated sprints to limit the development of peripheral fatigue beyond  
340 a critical threshold. *Experimental Physiology* 99: 951-963, 2014.
- 341 18. Ispirlidis I, Fatouros IG, Jamurtas AZ, Nikolaidis MG, Michailidis I, Douroudos I,  
342 Margonis K, Chatzinikolaou A, Kalistratos E, Katrabasas I, Alexiou V, and Taxildaris  
343 K. Time-course of changes in inflammatory and performance responses following a  
344 soccer game. *Clinical journal of sport medicine : official journal of the Canadian*  
345 *Academy of Sport Medicine* 18: 423-431, 2008.
- 346 19. Jones DA. High- and low-frequency fatigue revisited. *Acta Physiologica Scandinavica*  
347 156: 265-270, 1996.
- 348 20. Juel C. Muscle pH regulation: role of training. *Acta Physiologica Scandinavica* 162:  
349 359-366, 1998.
- 350 21. Keeton RB and Binder-Macleod SA. Low-Frequency Fatigue. *Physical Therapy* 86:  
351 1146-1150, 2006.
- 352 22. Kent-Braun AJ. Central and peripheral contributions to muscle fatigue in humans  
353 during sustained maximal effort. *Eur J Appl Physiol Occup Physiol* 80: 57-63, 1999.
- 354 23. Krstrup P, Ermidis G, and Mohr M. Sodium bicarbonate intake improves highintensity  
355 intermittent exercise performance in trained young men. *Journal of the International*  
356 *Society of Sports Nutrition* 12: 1-7, 2015.
- 357 24. Magalhães J, Rebelo A, Oliveira E, Silva JR, Marques F, and Ascensão A. Impact of  
358 Loughborough Intermittent Shuttle Test versus soccer match on physiological,  
359 biochemical and neuromuscular parameters. *European journal of applied physiology*  
360 108: 39, 2009.
- 361 25. McInnes SE, Carlson JS, Jones CJ, and McKenna MJ. The physiological load imposed  
362 on basketball players during competition. *Journal of Sports Sciences* 13: 387-397, 1995.
- 363 26. McNaughton LR, Siegler J, and Midgley A. Ergogenic Effects of Sodium  
364 Bicarbonate. *Current Sports Medicine Reports* 7: 230-236, 2008.
- 365 27. Millet GY, Martin V, Martin A, and Vergès S. Electrical stimulation for testing  
366 neuromuscular function: from sport to pathology. *Eur J Appl Physiol* 111: 2489-2500,  
367 2011.
- 368 28. Narazaki K, Berg K, Stergiou N, and Chen B. Physiological demands of competitive  
369 basketball. *Scand J Med Sci Sports* 19: 425-432, 2009.
- 370 29. Perrey S, Racinais S, Saimouaa K, and Girard O. Neural and muscular adjustments  
371 following repeated running sprints. *Eur J Appl Physiol* 109: 1027-1036, 2010.

- 372 30. Place N, Maffiuletti NA, Martin A, and Lepers R. Assessment of the reliability of  
373 central and peripheral fatigue after sustained maximal voluntary contraction of the  
374 quadriceps muscle. *Muscle Nerve* 35: 486-495, 2007.
- 375 31. Price M, Moss P, and Rance S. Effects of sodium bicarbonate ingestion on prolonged  
376 intermittent exercise. *Med Sci Sports Exerc* 35: 1303-1308, 2003.
- 377 32. Racinais S, Girard O, Micallef JP, and Perrey S. Failed Excitability of Spinal  
378 Motoneurons Induced by Prolonged Running Exercise. *Journal of Neurophysiology* 97:  
379 596-603, 2007.
- 380 33. Ratel S, Williams CA, Oliver J, and Armstrong N. Effects of Age and Recovery  
381 Duration on Performance During Multiple Treadmill Sprints. *Int J Sports Med* 27: 18,  
382 2006.
- 383 34. Saunders B, Sale C, Harris RC, and Sunderland C. Effect of sodium bicarbonate and  
384 Beta-alanine on repeated sprints during intermittent exercise performed in hypoxia. *Int*  
385 *J Sport Nutr Exerc Metab* 24: 196-205, 2014.
- 386 35. Siegler JC and Marshall P. The effect of metabolic alkalosis on central and peripheral  
387 mechanisms associated with exercise-induced muscle fatigue in humans. *Experimental*  
388 *physiology* 100: 519-530, 2015.
- 389 36. Siegler JC, Marshall P, Pouslen MK, Nielsen N-PB, Kennedy D, and Green S. The  
390 effect of pH on fatigue during submaximal isometric contractions of the human calf  
391 muscle. *Eur J Appl Physiol* 115: 565-577, 2015.
- 392 37. Siegler JC, Marshall PW, Bishop D, Shaw G, and Green S. Mechanistic Insights into  
393 the Efficacy of Sodium Bicarbonate Supplementation to Improve Athletic Performance.  
394 *Sports Med Open* 2: 11, 2016.
- 395 38. Siegler JC, Marshall PWM, Raftery S, Brooks C, Dowswell B, Romero R, and Green S.  
396 The differential effect of metabolic alkalosis on maximum force and rate of force  
397 development during repeated, high-intensity cycling. *J Appl Physiol (1985)* 115: 1634-  
398 1640, 2013.
- 399 39. Siegler JC, Mudie K, and Marshall P. The influence of sodium bicarbonate on maximal  
400 force and rates of force development in the triceps surae and brachii during fatiguing  
401 exercise. *Experimental physiology* 101: 1383-1391, 2016.
- 402 40. Sostaric SM, Skinner SL, Brown MJ, Sangkabutra T, Medved I, Medley T, Selig SE,  
403 Fairweather I, Rutar D, and McKenna MJ. Alkalosis increases muscle K<sup>+</sup> release, but  
404 lowers plasma [K<sup>+</sup>] and delays fatigue during dynamic forearm exercise. *The Journal*  
405 *of physiology* 570: 185-205, 2006.
- 406 41. Sparks A, Williams E, Robinson A, Miller P, Bentley DJ, Bridge C, and Mc Naughton  
407 LR. Sodium bicarbonate ingestion and individual variability in time-topeak pH.  
408 *Research in Sports Medicine* 25: 58-66, 2017.
- 409 42. Taylor JL and Gandevia SC. A comparison of central aspects of fatigue in submaximal  
410 and maximal voluntary contractions. *J Appl Physiol (1985)* 104: 542550, 2008.
- 411 43. Thomas K, Dent J, Howatson G, and Goodall S. Etiology and Recovery of  
412 Neuromuscular Fatigue following Simulated Soccer Match-Play. *Medicine & Science*  
413 *in Sports & Exercise* In Press, 2017.
- 414 44. Westerblad H. Acidosis Is Not a Significant Cause of Skeletal Muscle Fatigue. *Med Sci*  
415 *Sports Exerc* 48: 2339-2342, 2016.

416

417 **Figure Legends:**

418 Figure 1: Schematic of one quarter of the modified Loughborough Intermittent Sprint Test  
419 (LIST).

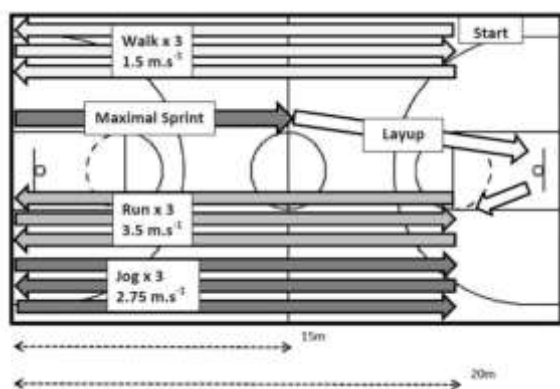
Figure 2: Neuromuscular function assessment by playing quarter. Data presented in mean  $\pm$  SD. A: MVIC force throughout the protocol; B: 100 Hz twitch force; C: 100 Hz twitch force. Significant group effect; \$ Significant time effect; \*Significant interaction effect

420

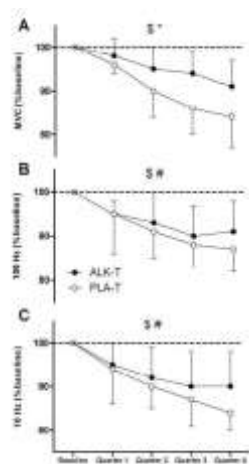
## TABLES

Table 1: Assessment of neuromuscular fatigue and performance indicators at all stages of both conditions (ALK-T: Alkalosis trial; PLA-T: Placebo trial). # Significant group effect; \$ Significant time effect; \* Significant interaction effect. All  $P < 0.05$ . All data is presented in mean  $\pm$  SD

Variable		Condition	Baseline	Quarter 1	Quarter 2	Quarter 3	Quarter 4
MVC	\$,*	ALK-T	255 $\pm$ 36	251 $\pm$ 40	244 $\pm$ 42	240 $\pm$ 43	233 $\pm$ 42
(N.m)		PLA-T	259 $\pm$ 32	247 $\pm$ 35	233 $\pm$ 41	223 $\pm$ 38	220 $\pm$ 42
100 Hz	#,\$	ALK-T	72 $\pm$ 7	69 $\pm$ 7	67 $\pm$ 9	65 $\pm$ 8	65 $\pm$ 6
(N.m)		PLA-T	70 $\pm$ 8	67 $\pm$ 10	64 $\pm$ 8	62 $\pm$ 9	61 $\pm$ 9
10 Hz	#,\$	ALK-T	71 $\pm$ 8	67 $\pm$ 8	65 $\pm$ 8	63 $\pm$ 6	63 $\pm$ 6
(N.m)		PLA-T	70 $\pm$ 7	66 $\pm$ 9	64 $\pm$ 8	61 $\pm$ 6	59 $\pm$ 7
10:100 Hz		ALK-T	98 $\pm$ 5	98 $\pm$ 3	97 $\pm$ 4	98 $\pm$ 3	96 $\pm$ 2
Ratio (%)		PLA-T	100 $\pm$ 5	99 $\pm$ 4	100 $\pm$ 3	100 $\pm$ 8	97 $\pm$ 5
15m Sprint	\$	ALK-T		2.53 $\pm$ 0.11	2.56 $\pm$ 0.12	2.58 $\pm$ 0.14	2.58 $\pm$ 0.11
(s)		PLA-T		2.54 $\pm$ 0.11	2.55 $\pm$ 0.13	2.58 $\pm$ 0.17	2.60 $\pm$ 0.16
Layup (%)		ALK-T		92.7 $\pm$ 7.2	82.7 $\pm$ 12.5	86.4 $\pm$ 13.0	87.3 $\pm$ 9.8
		PLA-T		88.2 $\pm$ 4.4	89.1 $\pm$ 5.7	85.4 $\pm$ 7.7	86.4 $\pm$ 10.7



ACCEPTED



ACCEPTED